

**PREVALENCE OF MICROALBUMINURIA
AND IT'S CORRELATION WITH DURATION AND SEVERITY OF
HYPERTENSION**

**Dissertation submitted for
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CERTIFICATE

This is to certify that this dissertation titled **PREVALENCE OF MICROALBUMINURIA AND ITS CORRELATION WITH DURATION AND SEVERITY OF HYPERTENSION- 'CROSS SECTIONAL STUDY'** submitted by **DR.S.SATHISHKUMAR** to the faculty of General Medicine, The Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch I (General Medicine) is a bonafide research work carried out by him under our direct supervision and guidance.

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their speedy recovery and place this study as a tribute to them and to the numerous others likely affected.

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Introduction

1. INTRODUCTION

1.1MICROALBUMINURIA

Microalbuminuria has been defined as albumin excretion of 20 to 200 mcg / minute or 30 to 300 mg /24 hour in a 24 hour urinary sample. Anything above this level is called macroalbuminuria.

Microalbuminuria can also be defined in terms of the urinary albumin –to – creatinine ratio (UACR). A ratio greater than 30 mg /g in the first voided sample in the morning (clean, midstream) is considered abnormal.

TABLE-1.1 Urine assays for Albuminuria/Proteinuria

24-Hour Albumin (mg/24 h)

Albumin/Creatinine Ratio(mg/g)

Dipstick Proteinuria

24-Hour	Urine	Protein	(mg/24h)
----------------	--------------	----------------	-----------------

Normal

8-10

<30

-

<150

Microalbuminuria

30-300

30-300

-/Trace/1+

-

Proteinuria

>300

>300

Trace-3+

>150

Albuminuria detected by immunoturbidometric assay

Albumin represents 30–70% of the total protein excreted in the urine

Microalbuminuria - the finding of albumin in the urine not detectable by the urine dipstick precedes the decline in GFR and heralds renal and cardiovascular complications.

If the patient already has established proteinuria, then testing for microalbumin is not necessary. Antihypertensive treatment reduces albuminuria and retards its progression even in normotensive diabetic patients.

The prevalence of microalbuminuria in essential hypertension has been variably studied and quoted as between 10 and 40%. Many studies have shown positive association of microalbuminuria in essential hypertensive patients with severity of hypertension, particularly with systolic blood pressure and pulse pressure. While some studies show it correlates more with mean ambulatory pressure.

In people older than 45 years with stage II hypertension, microalbuminuria seems to be strongly associated with severe cardiovascular risk factors and target organ damage.

Microalbuminuria strongly predicts cardiovascular morbidity and mortality, clinical nephropathy and progression of renal disease in high risk population.

Microalbuminuria has been shown to be an independent risk factor for cardiovascular diseases in hypertensive patients. Several retrospective and

cross sectional studies reported that the prevalence of cardiovascular disease is significantly higher among hypertension with microalbuminuria than patients without microalbuminuria.

Most of the studies show that microalbuminuria in hypertension is not affected by age, body weight, smoking, alcohol or family history of hypertension.

Microalbuminuria has been proposed as a marker of generalized vascular disease with arterial endothelial dysfunction involved in the pathogenesis of atherothrombotic vascular disease.

Hypertension doubles the risk of cardiovascular diseases, including coronary heart disease (CHD), congestive heart failure (CHF), ischemic and hemorrhagic stroke, renal failure, and peripheral arterial disease.

In this background, cross sectional study of 60 patients with hypertension of various age categories and duration of hypertension were studied. Prevalence of microalbuminuria and its correlation with duration and severity of hypertension (target

organ damage) were studied in South Indian patients, especially in southern districts of Tamilnadu, who are attending the Government Rajaji Hospital, Madurai. The study was undertaken in the department of medicine, Government Rajaji Hospital, Madurai.

It is hoped that this study may help to prevent the dreaded complications of untreated hypertension in our own South Indian population, by early detection of microalbuminuria.

The study also stresses the importance of regular intake of anti hypertensive drugs and thereby preventing early morbidity and mortality.

Review of literature

2. Review of literature

2.1 DEFINITION

TABLE 2.1.STAGING HYPERTENSION-JNC 7

Blood Pressure (BP) Stage	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Normal	<120	<80
Prehypertension	120-139	80-89
Stage 1 hypertension	140-159	90-99
Stage 2 hypertension	≥ 160	≥ 100

Clinically, *hypertension might be defined as that level of blood pressure at which the institution of therapy reduces blood pressure–related morbidity and mortality.*

Calculation of seated BP is based on the mean of two or more readings on two separate office visits.

According to JNC (Joint National Committee) 7 report, in adults aged 18 years and above, systolic blood pressure of <120 mm of Hg and diastolic blood pressure of <80mm of Hg is normal. Systolic blood pressure of 120 – 139 mm of Hg and diastolic blood pressure of 80 – 89 mm of Hg is prehypertension.

In stage 1 hypertension, the systolic blood pressure is 140-159 mm Hg and diastolic blood pressure is 90-99 mm of Hg. In stage 2 hypertension, systolic blood pressure is ≥ 160 mm of Hg and diastolic blood pressure is ≥ 100 mm of Hg.

The prevalence of hypertension in India varies widely from 3.80 to 15.63%

in men and 2 to 15.38% women in the urban areas and from 1.57 to 6.93% in men and 2.38% to 8.81% in women in rural areas.

Hypertension is a major risk factor for myocardial ischaemia and infarction. Patients with hypertension are also more susceptible to silent ischaemia. Endothelial cell is probably both a victim and an active participant in the vascular changes, that leads to hypertension. Microalbuminuria reflects widespread vascular damage due to generalized endothelial dysfunction that initiates the atherosclerotic process.

Microalbuminuria has been proposed as a marker of endothelial dysfunction, as microalbuminuria has been related to elevated concentrations of accepted markers of endothelial dysfunction such as Von Willebrand factor, thrombomodulin and activated factor VII.

Microalbuminuria might lead to hemodynamic strain and instability which can then, start the atherosclerotic process and eventually leads to cardiovascular events such as congestive heart failure, acute coronary syndromes, myocardial infarction and stroke.

2.2 HOME AND AMBULATORY MONITORING

Ambulatory monitoring provides automated measurements of BP over a 24-hour period, while patients are engaging in their usual activities, including sleep. Prospective outcome studies in both treated and untreated patients have shown that ambulatory BP measurement predicts fatal and nonfatal myocardial infarction and stroke, better than standard office measurements. Recommended normal values include an average daytime BP below 135/85 mm Hg, night time BP below 120/70 mm Hg, and 24-hour BP below 130/80 mm Hg. Some experts have recommended a lower cutoff

value of 130/80 mm Hg as a more stringent definition of normal daytime BP.

2.3 PATHOPHYSIOLOGY OF MICROALBUMINURIA

2.3.1 Glomerular and tubular mechanisms

The intimate relationship between low level albumin excretion and vascular permeability makes urinary albumin excretion, highly sensitive to the presence of any inflammatory process, including cardiovascular disease. The kidney is ideally placed to amplify any small changes in systemic vascular permeability.

The glomeruli receive 25% of the cardiac output. Of the 70kg of albumin that pass through the kidneys every 24 hour, less than 0.01% reaches the glomerular ultrafiltrate (i.e, less than 7g / 24hour) and hence enters the renal tubules.

Almost all filtered albumin is reabsorbed by the proximal tubule via a high – affinity, low – capacity endocytotic mechanism, with only 10 – 30 mg / 24 hour appearing in the urine. Assuming that, 7g of albumin is filtered every 24 hour, a 1% increase in systemic vascular permeability in response to an inflammatory stimulus would result in an additional 70mg of albumin into the filtrate. Since tubular mechanisms for albumin reabsorption are near saturation, urinary albumin excretion would increase from a maximum of 30 to approximately 100mg / 24hour.

Glomerular permeability to albumin is dependent on endothelial charge selectivity as well as size selectivity. The negative charge conferred on the glomerular membrane by its constituent glycoproteins plays a role in restricting the permeability of

glomerular charge selectivity, and this is found in diabetic and non diabetic populations with microalbuminuria.

2.3.2 Protiens in normal urine

Charge and size selectivity normally prevent virtually all plasma albumin, globulins and other large molecular weight proteins from crossing the glomerular wall. However, if this barrier is disrupted, there can be leakage of plasma proteins into the urine. Smaller proteins <20 kda are freely filtered but are readily reabsorbed by the proximal tubule.

Normal individuals excrete <150 mg/d of total protein and <30 mg/d of albumin. The remainder of the protein in the urine is secreted by the tubules, (Tamm-Horsfall, IgA, and urokinase) represents small amounts of filtered β 2-microglobulin, apoproteins, enzymes and peptide hormones.

In addition to treatment of hypertension in general, the use of ACE inhibitors and ARBs in particular are associated with additional renoprotection. These salutary effects are mediated by reducing intraglomerular pressure and inhibition of angiotensin driven sclerosing pathways, in part through inhibition of TGF- β mediated pathways.

Hypertension more commonly accompanies microalbuminuria or macroalbuminuria in type 2 DM.

2.4 Blood pressure and microalbuminuria

Similarly, alterations in the fraction of plasma filtered by the glomerulus

due to changes in blood pressure and intraglomerular pressure regulation, may also result in relatively large changes in urinary albumin excretion. It is therefore not surprising, that several studies have shown a positive correlation between microalbuminuria and blood pressure, especially at

pressures < 150/90mmHg. (Pontremoli R, Sofia A, Tirotta A, Revera M, Nicotella C, Viazzi F et.al., Deckert T, Kofoed – Enevoldsen A, Videl P, Norgaard K, Andreassen HB, Feldt – Rasmussen B).

In addition, insertion polymorphism of the angiotensin – 1 converting enzyme (ACE) gene, leading to higher circulating activities of ACE and a predisposition to cardiovascular disease, has been shown to be associated with microalbuminuria as measured by the albumin / creatinine ratio (Kofoed – Enevoldsen A, Foyle WJ, Fernandez M, Ydkin JS.). ACE activates angiotensin II as part of the rennin – angiotensin – aldosterone axis regulating blood pressure, angiotensin II having a powerful vasoconstrictor action as well as stimulating aldosterone release.

Systemic blood pressure on the other hand, correlated with intraglomerular pressure and microalbuminuria (Rodicio J, Campo C, Ruilope L., Lydakis C, Lip GY). Moreover, systolic blood pressure is one of the most relevant determinants of microalbuminuria (Bianchi S, Bigazzi R, Campese VM). However, microalbuminuria continues to be an independent risk factor for cardiovascular diseases even after adjustment for blood pressure.

In hypertensive patients, microalbuminuria is related to salt sensitivity,

absence of a nocturnal dip in blood pressure and higher mean 24 hour blood pressure measurements. All of these factors have been related to a

high prevalence of cardiovascular disease in hypertensive population (Lydakis C, Lip GY., Morimoto A, Uzu T, Fujii T, et.al).

2.5 Factors that cluster with microalbuminuria

Microalbuminuria may be related to target organ damage by several biological pathways:

- Insulin resistance
- Central obesity
- Low levels of high – density lipoprotein cholesterol
- High triglyceride levels
- Systolic hypertension
- Lack of nocturnal dip in blood pressure on 24 hour monitoring
- Salt sensitivity
- Endothelial dysfunction
- Hypercoagulability
- Impaired fibrinolysis
- Renal dysfunction

2.6 Endothelial function

Over 20 years ago, Parving (Parving HH, Jensen M, Morgensen CE, Evrin PE.) showed that urinary albumin excretion correlated with blood

pressure and the transcapillary escape rate of radiolabelled albumin in patients with essential hypertension.

Increased systemic capillary permeability has also been linked with microalbumin in health, (Felt – Rasmussen B) and recent studies suggest that endothelial dysfunction may lead to impaired insulin action as well as to capillary leakage of albumin, features which may be linked to a predisposition to cardiovascular disease

(Yudkin JS) .

2.7 Clinical features

In uncomplicated hypertensive patients, it is almost always asymptomatic. A person may be unaware of the consequent progressive target organ damage for as long as 10 to 20 years. Symptoms often attributed to hypertension are headache, tinnitus, dizziness and fainting which may be observed as commonly as in the normotensive population.

2.8 Complications of hypertension

The higher the level of blood pressure, the more likely that various cardiovascular diseases will develop prematurely through acceleration of atherosclerosis, the pathological hallmark of uncontrolled hypertension. If untreated about 50% of hypertensive people die of coronary artery disease or congestive heart failure, about 33% of stroke, and 10 to 15% of renal failure.

Those with rapidly accelerating hypertension die more frequently of renal failure, as do those who are diabetic, once proteinuria or other evidence of nephropathy develops. Death is usually attributed to stroke or myocardial infarction, instead of to the hypertension that was largely responsible.

In general, the vascular complications of hypertension can be considered as either “hypertensive” or “atherosclerotic”. The former are more directly caused by increased blood pressure per se and can be prevented by lowering this level; the latter

have more multiple causations.

s. no	Hypertension	Atherosclerotic
1.	Accelerated – malignant phase	Coronary heart disease
2.	Hemorrhagic stroke	Sudden death
3.	Congestive heart failure	Arrhythmias
4.	Nephrosclerosis	Atherothrombotic stroke
5.	Aortic dissection	Peripheral vascular disease

TABLE 2.2 Vascular complications of hypertension

2.9 HEART-HYPERTENSION-MICROALBUMINURIA

Heart disease is the most common cause of death in hypertensive patients. Hypertensive heart disease is the result of structural and functional adaptations, leading to left ventricular hypertrophy, diastolic dysfunction, CHF, abnormalities of blood flow due to atherosclerotic coronary artery disease and microvascular disease, and cardiac arrhythmias.

Both genetic and hemodynamic factors contribute to left ventricular hypertrophy. Clinically, left ventricular hypertrophy can be diagnosed by electrocardiogram, although echocardiography provides a more sensitive measure of left ventricular wall thickness. Individuals with left ventricular hypertrophy are at increased risk for CHD, stroke, CHF, and sudden death. Aggressive control of hypertension can regress or reverse left ventricular hypertrophy and reduce the risk of cardiovascular disease.

It has been acknowledged that microalbuminuria is a new marker of cardiologist. Nephrologists and diabetologists have traditionally measured microalbuminuria in their patients to monitor the development and progression of kidney disease, but now studies such as HOPE trial have shown a clear relationship between microalbuminuria and cardiovascular events.

In the MONICA study, one of the largest longitudinal studies to investigate predictive role of microalbuminuria, hypertensive subjects with albuminuria showed a four-fold raised risk of ischaemic heart diseases compared with hypertensive subjects without albuminuria.

2.10 BRAIN-HYPERTENSION- MICROALBUMINURIA

Hypertension is an important risk factor for brain infarction and hemorrhage. Approximately 85% of strokes are due to infarction and the remainder is due to

hemorrhage, either intracerebral hemorrhage or subarachnoid hemorrhage. The incidence of stroke rises progressively with increasing blood pressure levels, particularly systolic blood pressure in individuals >65 years. Treatment of hypertension convincingly decreases the incidence of both ischemic and hemorrhagic strokes. Hypertension is also associated with impaired cognition in an aging population, and longitudinal studies support an association between mid-life hypertension and late-life cognitive decline. Hypertension-related cognitive impairment and dementia may be a consequence of a single infarct due to occlusion of a "strategic" larger vessel or multiple lacunar infarcts due to occlusive small vessel disease resulting in subcortical white matter ischemia.

Several clinical trials suggest that antihypertensive therapy has a beneficial effect on cognitive function, although this remains an active area of investigation.

Cerebral blood flow remains unchanged over a wide range of arterial pressures (mean arterial pressure of 50–150 mmHg) through a process termed auto regulation of blood flow. In patients with the clinical syndrome of

malignant hypertension, encephalopathy is related to failure of autoregulation of cerebral blood flow at the upper pressure limit, resulting in vasodilation and hyperperfusion. Signs and symptoms of hypertensive encephalopathy may include severe headache, nausea and vomiting (often of a projectile nature), focal neurologic signs, and alterations in mental status. Untreated, hypertensive encephalopathy may progress to stupor, coma, seizures, and death within hours.

It has been shown that hypertensive patients with microalbuminuria have an increased thickness of the carotid intima and media layers suggesting a greater degree of atherosclerosis.

2.11 RENAL VASCULAR INJURY -MICROALBUMINURIA

Whether it is "essential" or of known etiology, hypertension results in development of intrinsic lesions of the renal arterioles (hyaline arteriosclerosis) that eventually lead to loss of function (nephrosclerosis).

Arteriolar nephrosclerosis is seen in patients who are hypertensive (BP > 150/90 mmHg) for an extended period of time but whose hypertension has not progressed to a malignant form. Such patients, usually in the older age group, are often discovered to be hypertensive on routine physical examination or as a result of nonspecific symptomatology (e.g., headaches, weakness, and palpitations).

The characteristic pathology is in the afferent arterioles, which have thickened walls due to deposition of homogeneous eosinophilic material (hyaline arteriosclerosis). Narrowing of vascular lumen leads to ischemic injury to glomeruli and tubules.

Physical examination may reveal changes in retinal vessels (arteriolar narrowing and/or flame-shaped hemorrhages), cardiac hypertrophy, and possibly signs of congestive heart failure. Renal disease may manifest as a mild to moderate elevation of serum creatinine concentration, microalbuminuria, or proteinuria.

2.12 ROLE OF RAAS

The Renin angiotensin aldosterone system plays an important role in modulating the effects of microalbuminuria.

Angiotensinogen II activates AT 1 receptor resulting in the stimulation of oxidative stress, inflammatory mediators, proliferation, and other mechanisms that contribute to the progression of renal disease. It is clear that blockade of RAS results in decreased progression of renal disease.

A critical driving factor with both renal and wider cardiovascular pathologies is over activation of RAS. Agents that delay the progression of renal disease therefore are also likely to be cardio protective. These agents not only lessen the systemic consequences of renal dysfunction, but may have cardio protective effects by exerting beneficial on endothelia elsewhere in the body and within the heart.

Renal artery stenosis

Renal artery stenosis (RAS) is the partial or complete occlusion of the renal artery, resulting in ischemia to kidney and renovascular hypertension.

RAS should be considered in the setting of poorly controlled hypertension despite four or more medications, hypertension with asymmetric kidney sizes, flash pulmonary edema, acute renal failure upon initiation of ACE inhibitors or ARBs or the presence of abdominal bruit.

A stenosis greater than 70% is considered potentially significant. Diagnosis may be obtained by a Doppler ultrasound.

Normal kidney sizes and textures on renal ultrasound **and lack of proteinuria** reflect normal underlying renal parenchyma and a higher likelihood of renal function improving with revascularization.

Unilateral renal artery stenosis alone rarely results in significant decline in GFR, so the treatment goals are more focused on controlling hypertension with ACE inhibitors or ARBs.

Bilateral renal artery stenosis angioplasty with stenting and bypass is the treatment of choice.

2.13 PERIPHERAL VASCULAR DISEASE-HYPERTENSION

In addition to contributing to the pathogenesis of hypertension, blood vessels may be a target organ for atherosclerotic disease secondary to long-standing elevated blood pressure. Hypertensive patients with arterial disease of the lower extremities are at increased risk for future cardiovascular disease.

Although patients with stenotic lesions of the lower extremities may be asymptomatic, intermittent claudication is the classic symptom of PAD. This is characterized by aching pain in the calves or buttocks while walking that is relieved by rest.

The ankle-brachial index is a useful approach for evaluating PAD and is defined as the ratio of noninvasively assessed ankle to brachial (arm) systolic blood pressure. An ankle-brachial index < 0.90 is considered diagnostic of PAD and is associated with

>50% stenosis in at least one major lower limb vessel. Several studies suggest that an ankle-brachial index < 0.80 is associated with elevated blood pressure, particularly systolic blood pressure.

FUNDUS PICTURE OF HYPERTENSIVE RETINOPATHY
(Arteriole attenuation, cotton wool spots, flame shaped hemorrhages)

HYPERTENSION-RETINOPATHY

Grade 1	Thickening and tortuosity of arteries showing 'silver wiring' appearance
Grade 2	Grade I changes plus arteriovenous nicking
Grade 3	Grade II changes plus flame-shaped (superficial) haemorrhages and cotton wool exudates
Grade 4	Grade 3 changes plus papilloedema

TABLE 2.3 Keith and Wagner's grading of hypertensive retinopathy

Marked systemic hypertension causes sclerosis of retinal arterioles, splinter hemorrhages, focal infarcts of the nerve fiber layer (cotton-wool spots), and leakage of lipid and fluid (hard exudate) into the macula.

In hypertensive crisis, sudden visual loss can result from vasospasm of retinal arterioles and retinal ischemia. In addition, acute hypertension may produce visual loss from ischemic swelling of the optic disc.

Patients with acute hypertensive retinopathy should be treated by lowering the blood pressure. However, the blood pressure should not be reduced precipitously, because there is a danger of optic disc infarction from sudden hypoperfusion.

Secondary Causes of Systolic and Diastolic Hypertension

Renal

Parenchymal diseases,
Renal cysts (including polycystic kidney disease),
Renal tumors (including renin-secreting tumors),
Obstructive uropathy

Arteriosclerotic,
Fibromuscular dysplasia

Adrenal

Primary aldosteronism,
Cushing's syndrome,
17-hydroxylase deficiency,
11-hydroxylase deficiency,
11-hydroxysteroid dehydrogenase deficiency (licorice), pheochromocytoma

Aortic coarctation

Obstructive sleep apnea

Preeclampsia/eclampsia

Neurogenic

Psychogenic, diencephalic syndrome,
Familial dysautonomia,
Polyneuritis (acute porphyria, lead poisoning),
Acute increased intracranial pressure,
Acute spinal cord section

Miscellaneous endocrine

Hypothyroidism,
Hyperthyroidism,
Hypercalcemia,
Acromegaly

Medications

High-dose estrogens, adrenal steroids, decongestants, appetite suppressants,
Cyclosporine, tricyclic antidepressants,
monamine oxidase inhibitors,
Erythropoietin,
Nonsteroidal anti-inflammatory agents, cocaine

Mendelian forms of hypertension

2.14. Guidelines for measuring blood pressure

a. *Posture*

1. Sitting postures are usually adequate for routine follow – up. Patient should sit quietly with back supported for five minutes and arm supported at the level of heart.

2. For patients who are over 65, diabetic or receiving anti hypertensive therapy, check for postural changes by taking readings immediately and 2 minutes after the patient stands.

b. *Circumstances*

No caffeine for preceding hour.

No smoking for preceding 15 minutes.

No exogenous adrenergic stimulants like phenylephrine in nasal decongestants or eye drops for pupillary dilation.

A quite, warm setting.

Home readings taken under various circumstances 24 hour ambulatory recordings may be preferable.

C. EQUIPMENT

C. *Cuff Size*

The bladder should encircle and cover 2/3 of arm length.

If not place the bladder over the brachial artery, if bladder is too small, spuriously high readings may result.

d. *Manometer*

Aneroid gauges should be calibrated every six months against the mercury manometer. For infants, use ultrasound equipment. e.g. The Doppler method.

e. *Technique*

i. *Number of readings*

On each occasion, take atleast two readings, separated by as much time as practical. If reading varies by more than 5 mm Hg, take additional readings until two are close. For diagnosis, obtain atleast three sets of readings a week apart.

Initially, take pressure in both arms, if pressure differs, use arm with higher pressure. If arm pressure is elevated, take pressure in one leg, particularly in patients below age 30.

ii. *Performance*

Inflate the bladder quickly to a pressure 20 mm Hg above the systolic, as recognized by disappearance of the radial pulse.

Deflate the bladder 3 mm Hg every second.

Record the Korotkoff phase V(disappearance) except in children,in whom use of phase IV (muffling) is advocated.

If korotkoff sounds are weak, have the patients raise the arm, open and close the hand 5 to 10 times, after which the bladder should be inflated quickly.

f. Recording

Note the blood pressure, patient's position, and the arm, cuff size (eg.140/90, seated, right arm, and large adult cuff)

2.15 SCREENING FOR MICROALBUMINURIA

ADA-American Diabetes Association recommended methods for measuring microalbuminuria

Albumin to creatinine ratio in a random spot collection

24- hour collection with creatinine

Timed urine collection over four hours or overnight

ADA advises that the first method is often easy to carry out in the office setting and generally provides accurate information.The first void urine or other morning collections are best because of diurnal variations in albumin secretion.

2.16 Interventions to reduce microalbuminuria

Endothelial dysfunction occurs early in patients with microalbuminuria. Several large

studies show modulating microalbuminuria has beneficial effects.

Non pharmacological measures

These include weight loss, exercise and eating a low fat diet, most of the time these is however not enough.

Pharmacological agents

Statins, ACE inhibitors, ARBs have been shown in several land mark studies to decrease high blood pressure and microalbuminuria.

Either ACE inhibitors or ARBs should be used to reduce the progression from microalbuminuria to macroalbuminuria and the associated decline in GFR that accompanies macroalbuminuria in individuals with “Hypertension”. Although direct comparisons of ACE inhibitors and ARBs are lacking, most experts believe that the two classes of drugs are equivalent. ARBs can be used as an alternative in patients who develop ACE inhibitor –associated cough or angioedema.

After 2–3 months of therapy in patients with microalbuminuria, the drug dose is increased until either the microalbuminuria disappears or the maximum dose is reached. If use of either ACE inhibitors or ARBs is not possible, then calcium channel blockers (non-dihydropyridine class), beta blockers, or diuretics should be used. However, their efficacy in slowing the fall in the GFR is not proven. Blood pressure control with any agent is extremely important, but a drug-specific benefit in nephropathy, independent of blood pressure control, has been shown only for ACE inhibitors and ARBs in patients with DM.

The ADA suggests modest restriction of protein intake in diabetic individuals with microalbuminuria (0.8 g/kg per day) or macroalbuminuria (<0.8 g/kg per day, which is the adult Recommended Daily Allowance, or ~10% of the daily caloric intake).

Nephrology consultation should be considered when the estimated GFR < 60 mL/min per 1.743 m². Once macroalbuminuria ensues, the likelihood of ESRD is very high.

Aims and objectives

AIMS AND OBJECTIVES OF THE STUDY

To study the prevalence of microalbuminuria in patients with hypertension.

To study whether microalbuminuria correlates with duration of hypertension.

To study whether microalbuminuria correlates with severity of hypertension-JNC 7 staging.

To study whether microalbuminuria correlates with severity of Target organ damage (TOD).

Materials and methods

MATERIALS AND METHODS

SETTING

Outpatient clinic of Government Rajaji Hospital, Madurai.

DESIGN OF STUDY

Analytical-cross sectional study

PERIOD OF STUDY

Six months (August 2008-January 2009)

SAMPLE SIZE AND SELECTION OF STUDY SUBJECTS

Sixty hypertensive patients (both newly and previously diagnosed) attending the outpatient clinic were included in this Analytical-cross sectional study. This study group included 33 males and 27 females. The subjects included in the age groups ranging between 20yrs and 70yrs.

DETAILS OF STUDY SUBJECTS

Blood pressure was recorded using sphygmomanometer with standard cuff on 2 occasions 10 minutes apart. Patients should have refrained from smoking or drinking tea or coffee for at least 30 minutes before measuring BP. The higher of the two readings was taken as the patient's blood pressure.

Patients' height and weight were measured and the body mass index was calculated using the formula weight/height^2 . All the peripheral pulses were checked with special attention to carotids and femorals, to detect evidence for early atherosclerosis. An ocular fundus examination was done to detect hypertensive retinopathy.

All the subjects had routine urine analysis (albumin, sugar and deposits) done. Fasting and 2 hour postprandial blood sugar, serum urea and creatinine were estimated. A 12 lead electrocardiogram and chest X-ray were also taken.

All the patients were instructed to report with first early morning urine sample. Albumin in the urine is very stable at normal temperature, so urinary samples need not be frozen when sent to laboratory.

Microalbuminuria was detected by the immunoturbidometric assay.

Procedure of the test

All samples were tested on the morning of the visit to the clinic.

Microalbuminuria was detected by the immunoturbidometric assay. It is a fully

automated calibrated system. **Urine Albumin to creatinine ratio** in a

random spot collection is calculated.

Albumin

Technology - fully automated immunoturbidometric assay.

Method - photometry

Reference - adults range < 18mcg/ml

Creatinine

Technology - JAFFE Method - Rate Blanked and compensated

Method - photometry

Reference range: a) Males - 39 - 259 mg/dl

b) Females - 28 - 217 mg/dl

Exclusion criteria

Diabetes mellitus

Microalbuminuria detected by dipstick (Trace–3+)

Obesity

Febrile patients

Patients with UTI

Patients on ACE inhibitors

Ethical approval

Ethical committee clearance was obtained.

Consent

Informed consent was obtained from the subjects.

Statistical analysis

Computer analysis of data was done using a software epidemiological information package developed by the Centers for Disease control and prevention- Atlanta in collaboration with World Health Organization. Chi - square test is used for tests of significance.

Results and observation

TABLE NO 5.1: DISTRIBUTION OF VARIABLES IN THE STUDY GROUP

		FREQUENCY	PERCENTAGE	CUMULATIVE
A. AGE	20-30	2	3.3	3.3
	30-40	6	10.0	13.3
	40-50	20	33.3	46.7
	50-60	16	26.7	73.3
	60-70	11	18.3	91.7
	>70	5	8.3	100
B. SEX	FEMALE	27	45	45
	MALE	33	55	100
C. ECG CHANGES	ABSENT	28	46.7	46.7
	PRESENT	32	53.3	100
D. LVH	ABSENT	33	55	55
	PRESENT	27	45	100
E. CAD/LVF	ABSENT	40	66.7	66.7
	PRESENT	20	33.3	100
F. RETINOPATHY	ABSENT	45	75	75
	PRESENT	15	25	100
G. NEPHROPATHY	ABSENT	53	88.3	88.3
	PRESENT	7	11.7	100
H. SECONDARY HT CHANGES	ABSENT	54	90.0	90.0
	PRESENT	6	10.0	100
I. MICRO- ALBUMINURIA	ABSENT	22	36.7	36.7
	PRESENT	38	63.3	63.3
J. SYSTOLIC HT (JNC 7)	Stage I HT(140-159)	31	51.7	51.7
	Stage II HT(\geq 160)	29	48.3	48.3
K. DIASTOLIC HT (JNC 7)	Stage I HT(90-99)	33	55.0	55.0
	Stage II HT(\geq 100)	27	45.0	45.0

The age, sex, and target organ damage frequency and percentage distribution is depicted in the above table.

Age category

TABLE NO. 5.2. CENTRAL TENDENCIES AND DISPERSION OF STUDY VARIABLES

↓VARIABLE	MEAN	MEDIAN	SD
A. AGE	53.30	51.5	11.9
B. DURATION	41.37	36	24.8
C. SYSTOLIC BP	167.33	160	20.5
D. DIASTOLIC BP	104.6	100	11.3
E. PULSE PRESSURE	63.4	60	18.01
F.MEAN ARTERIAL PRESSURE	125.5	123.3	11.8
G. MICROALBUMINURIA	92.1	93.6	68.1

There were a total of 60 cases. Out of 60 cases 38(63%) had microalbuminuria positive and 22(27%) tested negative for microalbuminuria .

In the study group

Age distribution at the interval of 10 years, between 20 years and 70 years and above was included. The mean age distribution in the study group is 53.30. The maximum number of cases 20 (33.33%) belongs to the 40-50 years category. The next large number of cases belongs to 50-60years category around 16(26.67%) cases. 11(18.33%) cases included in the 60-70 years , 6(10%) cases in 30-40 years and 5(8.3%) cases in the >70 years and 2(3.3%) cases in 20-30 years category. The p value is 0.121 which is insignificant.

The sex distributions of the 60 cases, 33(55%) cases are males and 27(45%) cases are females.

Table No.5.3: RELATIONSHIP BETWEEN DURATION OF HT AND MICROALBUMINURIA

		MAU STATUS				
		ABSENT		PRESENT		
		COUNT		COUNT		COUNT
DURATION	1-59 M	19	67.9%	9	32.1%	28
OF HT	60-119 M	3	9.7%	28	90.3%	31
	>120 M	0	.0%	1	100.0%	1
TOTAL		22	36.7%	38	63.3%	60

In this study group, cases were categorized into three groups according to the duration of hypertension (i) 1-59 months, (ii) 60-119 months, (iii) >120 months.

Twenty eight cases presented in 1-59 months category. Out of 28 cases, 9(32.1%) tested positive for microalbuminuria, 19(68%) tested negative for microalbuminuria. 31 cases presented in 60-119 months category, 28(90%) cases tested positive for microalbuminuria, only 3(10%) cases tested negative for microalbuminuria.

CORRELATION BETWEEN DURATION OF HT AND MICROALBUMINURIA

All cases in the category > 120 months tested positive for microalbuminuria (100%).

TABLE NO 5.4: CORRELATION BETWEEN DURATION OF HT AND MICROALBUMINURIA

		DURATION OF HT	MICRO ALBUMINURIA
DURATION OF HT	PEARSON CORRELATION	1	.677(**)
	SIG. (2-TAILED)		.000
	N	60	60
MICRO ALBUMINURIA	PEARSON CORRELATION	.677(**)	1
	SIG. (2-TAILED)	.000	
	N	60	60

CHI SQUARE TEST: $p < 0.001^*$

The p value is < 0.001 and is statistically significant and duration of hypertension significantly correlates with microalbuminuria.

This study indicates that longer the duration of hypertension more number of cases are microalbuminuric and is statistically significant ($p < 0.001$).

TABLE NO.5.5: RELATIONSHIP BETWEEN SYSTOLIC BP AND MICROALBUMINURIA.

			MICROALBUMINURIA		TOTAL	CHI SQUARE
			ABSENT	PRESENT		
SYSTOLIC BP	STAGE I	HT(140-159)	17	6	23	$P < .001$
	STAGE II	HT(≥ 160)	5	32	37	
TOTAL			22	38	60	

The mean systolic blood pressure in the study group is 167 mm of Hg.

Among the study group

23(38%) cases were in stage I Hypertension and 37(62%) cases were in stage II hypertension.

6/23(26%) cases in stage I hypertension were tested positive for microalbuminuria. 17/23(74%) cases were tested negative for microalbuminuria.

32/37(86%) cases in stage II hypertension tested positive for microalbuminuria. 5/37(14%) cases tested negative for microalbuminuria.

TABLE NO: 5.6: CORRELATION BETWEEN SYSTOLIC BP AND MICROALBUMINURIA

	MICRO
BP-SYS	ALBUMINURIA

BP-SYS	PEARSON	1	.594(**)
	CORRELATION		
	SIG.(2-TAILED)		P<.001
	N	60	60
MICRO ALBUMINURIA	PEARSON	.594(**)	1
	CORRELATION		
	SIG. (2-TAILED)	P<.001	
	N	60	60

The p value is<0.001, which is statistically significant and correlates with microalbuminuria.

TABLE NO: 5.7: RELATIONSHIP BETWEEN DIASTOLIC BP AND MICROALBUMINURIA

		MICROALBUMINURIA		TOTAL	CHI SQUARE
		ABSENT	PRESENT		
DIASTOLE BP JNC7	STAGE I HT(90-100)	7	6	13	P=0.197
	STAGE II HT(>100)	15	32	47	
TOTAL		22	38	60	

The mean diastolic blood pressure in the study group is 105 mmHg. Among the study group, 13 cases were in stage I hypertension and 47 cases in stage II hypertension. 6/13 in diastolic stage I hypertension tested positive for microalbuminuria.

32/47 cases tested positive for diastolic stage II hypertension. The p value is 0.197, which is statistically insignificant, and does not correlate with severity.

TABLE NO: 5.8: RELATIONSHIP BETWEEN PULSE PRESSURE AND MICROALBUMINURIA

		MICROALBUMINURIA		TOTAL	CHI SQUARE
		ABSENT	PRESENT		
PULSE PRESSURE	30-59	12	10	22	P=0.032*
	60-90	10	22	32	
	>90	0	6	6	
TOTAL		22	38	60	

The mean pulse pressure in study group is 63 mm of Hg.

The pulse pressure category is divided into three groups. In the pulse pressure category 30-59 mm of Hg, 22 cases were present and among 22 cases, 10.(45.5%) tested

positive for microalbuminuria. In the pulse pressure category 60-90 mm of Hg, 32 cases were tested positive and among 32 cases, 22(68.8%) cases tested positive for microalbuminuria. In the pulse pressure category >90 mm of Hg, 6 cases were present and all (100%) cases tested positive for microalbuminuria.

TABLE NO: 5.9: CORRELATION BETWEEN PULSE PRESSURE AND MICROALBUMINURIA

		PULSE PRESSURE	MICRO ALBUMINURIA
PULSE PRESSURE	PEARSON CORRELATION	1	.447(**)
	SIG. (2-TAILED)		P<0.001
	N	60	60
MICRO ALBUMINURIA	PEARSON CORRELATION	.447(**)	1
	SIG. (2-TAILED)	.000	
	N	60	60

TABLE NO. 5.10: RELATIONSHIP BETWEEN MICROALBUMINURIA AND ECG CHANGES

		ECG CHANGE		TOTAL	CHI SQUARE P=0.183
		ABSENT	PRESENT		
MICROALBUMINURIA	ABSENT	13	9	22	
	PRESENT	15	23	38	
TOTAL		28	32	60	

23/32(72%) cases which presented with ECG changes tested positive for microalbuminuria. The p value is 0.183, which is statistically not significant.

LVH TOTAL CHI

MICROALBUMINURIA		ABSENT	PRESENT		SQUARE
	ABSENT	15	7	22	0.178
	PRESENT	18	20	38	
TOTAL		33	27	60	

TABLE.NO:5.11: RELATIONSHIP BETWEEN MICROALBUMINURIA AND LVH CHANGES

In the study group, 27 cases presented with LVH. Out of 27, 20(74%) cases tested positive for microalbuminuria. The p value is 0.178, which is not significant.

TABLE NO: 5.12: RELATIONSHIP BETWEEN MICROALBUMINURIA AND CAD/LVF

MICROALBUMINURIA		CAD/ LVF		TOTAL	CHI SQUARE
		ABSENT	PRESENT		
	ABSENT	18	4	22	0.09
	PRESENT	22	16	38	
TOTAL		40	20	60	

In this study 20 cases were presented with coronary artery diseases, left ventricular failure. Out of 20 cases 16 tested positive for microalbuminuria.

The p value is 0.09, which is less significant.

TABLE NO.5.13: RELATIONSHIP BETWEEN MICROALBUMINURIA AND RETINOPATHY

		RETINOPATHY		TOTAL	CHI SQUARE
		ABSENT	PRESENT		
MICROALBUMINURIA	ABSENT	22	0	22	P<001**
	PRESENT	23	15	38	
TOTAL		45	15	60	

In this study,15 cases presented with retinopathy all cases tested positive for microalbuminuria(100%).The p value is <0.001 and is statistically significant.

In this study, microalbuminuria correlates significantly with retinopathy.

TABLE 5.14: RELATIONSHIP BETWEEN MICROALBUMINURIA AND NEPHROPATHY

		NEPHROPATHY		TOTAL	CHI SQUARE
		ABSENT	PRESENT		
MICROALBUMINURIA	ABSENT	22	0	22	P<.033**
	PRESENT	31	7	38	
TOTAL				60	
		53	7		

Out of 7 cases, all 7 cases tested positive for microalbuminuria (100%).The p value is 0.03, and is statistically significant.

TABLE NO.5.15: RELATIONSHIP BETWEEN MICROALBUMINURIA AND SECONDARY HT

		SECONDARY HT		TOTAL	CHI SQUARE
		ABSENT	PRESENT		
MICROALBUMINURIA	ABSENT	25	0	25	0.029**
	PRESENT	29	6	35	
TOTAL		54	6		

In this study, 6 cases presented with secondary hypertension. 2 cases were pheochromocytoma, 2 cases were renal artery stenosis and 2 cases of coarctation of aorta. All 6(100%) cases tested positive for microalbuminuria.

The p value is 0.029 and is statically significant.

TABLE NO 5.16: RELATIONSHIP BETWEEN MICROALBUMINURIA AND CVA

		CVA		TOTAL	CHI SQ
		ABSENT	PRESENT		
MAU STATUS	ABSENT	20	2	22	P=0.229*
	PRESENT	29	9	38	
	TOTAL	49	11	60	

In this study, 11 cases presented with cerebrovascular accident. Out of 11 cases of CVA, 9(82%) cases tested positive for microalbuminuria. Only 2(18%) cases tested negative for microalbuminuria.

The P value is 0.229, which is less significant.

TABLE NO 5.16: RELATIONSHIP BETWEEN MICROALBUMINURIA AND TARGET ORGAN DAMAGE
TOD **CHI SQ**

		ABSENT	PRESENT	TOTAL	P=0.024*
MAU STATUS	ABSENT	12	10	22	
	PRESENT	9	29	38	
TOTAL		21	39	60	

In this study, 29(74%) out of 39 cases presented with target organ damage were tested positive for microalbuminuria.

The p value is 0.024 and is correlated statistically significant.

Discussion and comparative analysis

DISCUSSION AND COMPARATIVE ANALYSIS

In the present study, out of sixty cases of hypertension 38 cases had microalbuminuria. The prevalence of microalbuminuria in hypertension in this study shows 63.33%. This study has high prevalence of microalbuminuria.

In an Indian study conducted by kothiaiwale, Jagadeesh, Preethy, Verghesse, Solbannvara., KLE hospital Belgam the prevalence of microalbuminuria was 48%. In another Indian study conducted by V.K. Sharma, T.N Dubey, R.K. Jain., Gandhi hospital Bhopal, prevalence of microalbuminuria was 24% and had a significant correlation with severity of hypertension. It showed that cases having less than 10 yrs duration of hypertension, only 32% had microalbuminuria whereas cases having more than 10 yrs duration of hypertension all cases (100%) had microalbuminuria. This study shows that among cases having less than 5 yrs duration of hypertension 9/28(32.1%) cases had Microalbuminuria. Among cases between 5&10 yrs duration of hypertension 28/31(90.3%) cases had microalbuminuria. Among cases having more than 10 yrs duration of hypertension all (100%) had microalbuminuria.

This study indicates that longer the duration of hypertension more number of cases are microalbuminuric and is statistically significant ($p < 0.001$).

In the clinical epidemiology study conducted by John Forman and Naomi, showed increasing microalbuminuria even within normal range which is considered normal, is independently associated with an increased risk for development of hypertension.

Likewise severity of hypertension had significant correlation with microalbuminuria, among 38 cases who presented with microalbuminuria. Palatine p. et al in their study on 870 never treated hypertensives, found that family history of hypertension, smoking, alcohol, coffee intake and physical activity habits did not influence albumin excretion rate.

Convincing evidence has shown association of elevated urinary albumin excretion in hypertension with higher systolic blood pressure and pulse pressure through pressure dependant and non pressure dependant mechanisms, overweight, glucose intolerance, other phenotypes of metabolic syndrome smoking, genetically determined elevated renin angiotensin II levels and/or activity.

Furthermore, microalbuminuria consistently showed independent connection with cardiovascular morbidity and mortality. Besides, sharing adverse cardiovascular and metabolic risk pattern, it may be characterized by hyperfiltration possibly conducive to glomerular hypertension and eventually renal insufficiency.

This study correlates systolic blood pressure, diastolic blood pressure, pulse pressure with microalbuminuria and is statistically very significant ($p < 0.001$) with systolic

blood pressure and pulse pressure .

In Parving HH et.al shows a positive correlation with systolic and pulse pressure. In creosola g.et.al study shows a positive correlation of diastolic blood pressure with microalbuminuria.

This study shows 6/23(26.08%) cases in stage I HT and 32/37(86.48%) cases in stage II HT tested positive for microalbuminuria.

The severity of hypertension significantly correlates with microalbuminuria ($P<0.001$).

In an Indian study conducted by kothiawale, Jagadeesh, Preethy, Verghesse, Solbannvara., KLE hospital Belgam 30% cases in stage I HT and 70% cases in stage II HT tested positive for microalbuminuria.

This study indicates that higher the numbers of systolic blood pressure values more number of cases are microalbuminuric.

This study shows a significant correlation between target organ damage with microalbuminuria and it shows 29/39 of TOD (74.35%) cases tested positive with microalbuminuria.

Among patients with target organ damage (TOD)

15/15 (100%) cases of Retinopathy tested positive for microalbuminuria.

7/7 (100%) cases of Nephropathy tested positive for microalbuminuria.

20/27 (74%) cases of LVH tested positive for microalbuminuria and 7(26%) cases tested negative for microalbuminuria.

16/20 (80%) cases of CAD/LVF tested positive for microalbuminuria and 4 cases tested negative for microalbuminuria.

9/11 (82%) cases of CVA tested positive for microalbuminuria and only 2/11(18%) cases tested negative for microalbuminuria.

In Agarwal et al. study of 11,343 non diabetic hypertensives, those with microalbuminuria had a significantly prevalence of higher prevalence of microalbuminuria ($P<0.001$) of coronary artery disease (31% vs 22%),left ventricular hypertrophy(24% Vs 14%), previous stroke (6% Vs 4%),peripheral vascular disease(7% Vs 5%).

In an Indian study conducted by kothiawale, Jagadeesh, Preethy, Verghesse, Solbannvara., KLE hospital Belgam microalbuminuria showed a positive correlation with Retinopathy (100%), Nephropathy (100%), and LVH (85%), and 16/20 cases of CAD/LVF(65%),and 9/11 cases of CVA(85%).

This study included cases of secondary hypertension and all cases (100%) tested positive for microalbuminuria.

CLINICAL IMPLICATION

The prevention of hypertension is the major focus of the most recent JNC report which emphasizes that the individuals with the systolic blood pressure of > 120 mm of Hg or diastolic blood pressure > 80 mm of Hg are at risk of progressing to risk of overt hypertension. The size of prehypertension category is considered high. It would be useful for clinician to have a biochemical test that could aid in the identification of

individuals, most likely to develop hypertension. URINE ALBUMIN CREATININE RATIO (UACR) is a candidate for such a marker and it is comparable to other markers such as C-reactive protein, assessed for prediction of cardiovascular events in healthy populations.

Large studies confirmed that very low degrees of urine albumin excretion below the conventional threshold for microalbuminuria (UACR of 30 mg/gm) as low as 1.7 to 3.8 mg/gm for men and 3.4-7.5 mg/gm for women were associated with 71% increase in risk for developing hypertension.

Low grade albumin excretion has also been associated with risk of cardiovascular events in both diabetic and non-diabetic individuals. Many studies support to the notion that conventional microalbuminuria may be higher than the cut point at the substantial risk for adverse outcomes begins to be detectable.

In this study, microalbuminuria had a stronger association with target organ damage and therefore, screening for microalbuminuria in hypertensive patients is recommended as a cost effective biochemical tool in detecting future adverse outcomes, and thereby pharmacological and non pharmacological interventions can be made at the earliest.

In a recent study by Lazzara and Deen suggested, that states of glomerular hyperfiltration or increased activity of the renin angiotensin system of the kidney may provide a link between higher levels of albumin excretion and the development of hypertension.

European guidelines for Hypertension emphasize the importance of assessing the importance of target organ damage for cardiovascular risk stratification. Non invasive assessment of cardiac and peripheral arterial structures by ultrasonography is of relatively high cost and hence not performed routinely, recommended in selected cases.

Optimizing the diagnostic approach to the detection of target organ damage is utmost importance for a rationale and cost effective allocation of economic resources.

Evaluation of microalbuminuria is recommended as a screening tool for target organ damage especially in low risk patients in Hypertension.

Therefore Urinary screening for microalbuminuria *atleast once in a year in every hypertensive patients* improves the targeting of primary prevention and results in reduction in target organ damage.

Current challenges therefore should preclude the inclusion of microalbuminuria in the routine management of hypertensive patients.

Heightened awareness of microalbuminuria as an early prognostic indicator of target organ damage risk, knowing when, how, and in whom to screen for it, and finally, knowing the strategies to manage it are therefore essential prerequisites in therapeutic decision making process.

Conclusion

CONCLUSION

In this study of 60 patients with hypertension, the prevalence of microalbuminuria was found to be high.

The presence of microalbuminuria in hypertension is statistically correlated with the duration of hypertension.

This study showed statistically significant correlation with systolic blood pressure and pulse pressure, but not with diastolic pressure.

This study showed statistically significant correlation with severity of hypertension (JNC 7 STAGING).

This study showed statistically significant correlation with target organ damage.

.Therefore urinary screening for microalbuminuria in patients with hypertension improves the targeting of primary prevention and should result in the reduction of target organ damage.

PROFORMA

NAME: AGE: SEX:
ADDRESS: OCCUPATION:
DURATION OF HYPERTENSION: OP/IP NO. :
UNIT:
SYMPTOMS:
Head ache Polyuria Cold intolerance
Giddiness Oliguria Weight gain
Blurring of vision Puffiness of face Hoarseness of vision
Epistaxis Swelling of legs Centripetal obesity
Chest pain Anorexia Hirsutism
Palpitation Easy fatiguability Amenorrhoea/Menorrhagia
Dyspnoea Altered bowel habits Episodes of weakness
Fever Hematuria UTI

PAST HISTORY

DM
Renal disorders
Thyroid disorders

DRUG INTAKE

OCP
Corticosteroids

PERSONAL HISTORY

Smoking Alcoholism Drug abuse

FAMILY HISTORY

HT DM

ANTHROPOMETRY

Height (in cm): Weight (in kg): BMI:

GENERAL EXAMINATION

Fundus Xanthoma
Thyroid swelling Xanthelesma
Skin manifestations of thyroid disorders
Features of Cushing's syndrome

PULSE

Rate	Rhythm	Volume	Character
Radio femoral delay			Peripheral pulses

BLOOD PRESSURE (in mm hg)

	R	L	R	L
Sitting posture	UL		LL	
Lying posture	UL		LL	

SYSTEMIC EXAMINATION

CVS:

RS:

ABDOMEN:

CNS:

INVESTIGATIONS

Blood Glucose: Urea: Creatinine:

ECG: ECHO: Chest X Ray

URINE Albumin: Sugar: Deposits:

MICROALBUMINURIA

(Albumin creatinine Ratio)

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ABBREVIATION

ACE - Angiotensin converting enzyme

ARB - Angiotensin receptor blockade

AT 1 – Angiotensin I receptor

BP – Blood pressure

CAD – Coronary heart Disease

CVA-Cerebro Vascular Accident

ECG – Electrocardiogram

DM – Diabetes Mellitus

HT – Hypertension

JNC – Joint national committee

LVH – Left Ventricular Hypertrophy

MAU -microalbuminuria

PAD – peripheral arterial disease

RAS – Renin angiotensin system

TGF- β – transforming growth factor

TOD – Target organ damage

UTI – Urinary tract infection

UACR – Urinary albumin creatinine ratio